of IgG and binds complement via the classical and alternate pathology of complement. Acantholytic lesions typical of pemphigus can be produced in skin explants cultured in media enriched with pemphigus serum.

Several studies have shown that in most cases, during the acute phase of the disease, the titer of antibody in the serum correlates with the extent and severity of the disease. Therefore, patients without measurable antibody level have minimal disease while patients with high titers have widespread disease.

Controversy exists about the role and the benefit of following the antibody titer as a guide to therapy. Recent studies of clinical status in patients have shown that a rise in titer is associated with an exacerbation. Numerous cases are cited in the literature to indicate that clinical usefulness of sequential titers as a guide in therapy is still controversial. They feel that changes in clinical status are the best determinants of therapeutic maneuvers.

There are several reasons that can explain the discrepancy in results. The timing of the sequential serum specimens is not the same in all studies. None of the studies have shown correlation between *in vivo* tissue bound antibody and disease free state. Finally, in different studies different drugs have been used. The data become difficult to interpret because different immunosuppressive agents act differently and alter the immune response. Most studies have not used large enough numbers of patients for sound statistical analyses.

Whether the antibody titer remains a useful guide to monitor therapy for a group of patients is still uncertain. However, for a particular patient a twofold rise or fall in the antibody titer is significant, depending on the dynamics of the immune response. In most patients these changes occur simultaneously with changes in clinical status. In some patients the antibody changes occur before or after changes in clinical status.

The importance and the value of the pemphigus antibody cannot be denied. It is very specific for pemphigus and is present in virtually all cases. It may occasionally be absent due to species or organ specificity of the antibody, prozone phenomenon, interference with other antibodies or error in technique. Pemphigus-like antibodies, are seen transiently in low titers in patients with burns, fungal infections, systemic lupus erythematosus (SLE), myesthenia gravis, toxic epidermal necrolyses (TEN) and drug eruptions, and cross-react with blood group substances.

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Mucocutaneous Lymph Node Syndrome

THE MUCOCUTANEOUS LYMPH NODE SYNDROME (MLNS) was first described by Kawasaki in Japan. It has now been reported worldwide. MLNS almost exclusively affects prepubertal children with a peak incidence during the first 18 months with a slight male dominance.

The principal symptom is a spiking fever ranging from 38 to 40°C (100.4 to 104°F), lasting 5 to 20 days and unresponsive to antibiotics. In most cases there is bilateral congestion of the bulbar conjuctiva, dryness, erythema and fissuring of lips, protuberance of the lingual papillae and erythema of the oropharynx. The exanthem typically starts between the third and fifth day of illness and begins as a pronounced erythema of palms and soles with edema and a painful swelling causing limitation of movement. The rash spreads to the trunk in a variable pattern presenting as erythema multiforme, iris-colored lesions or a morbilliform or a scarletiniform eruption. During the second week, a desquamation begins starting at the junction of the skin and nail, extending to fingers, toes and subsequently to the remainder of the body. Significant frequent finding is a unilateral or bilateral nonsuppurative cervical lymphadenopathy of 1.5 cm or greater. Other infrequent clinical features are diarrhea, arthritis, mild hepatitis with jaundice, aseptic meningitis and carditis.

No specific laboratory test is diagnostic. Commonly, a leucocytosis with a left shift increased sedimentation rate and rarely elevated IgE is seen.

The mortality rate is about 1 to 2 percent. Death is frequent in children when cardiac com-

plications develop. Autopsy findings show that arteritis involving most major blood vessels is the primary histologic change. Identical lesions are seen in infantile polyarteritis nodosa. The cause is unknown at present.

MLNS must be clinically differentiated from scarlet fever, juvenile rheumatoid arthritis, systemic lupus erythematosus, erythema multiforme and childhood viral syndromes. There is no specific treatment. Some authors have suggested use of steroids while others recommend use of azathioprine with steroids.

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Langerhans Cell—Epidermal Macrophage

THE MAMMALIAN EPIDERMIS consists of three major cell types: the keratinocytes, responsible for keratinization; the malanocytes that produce and distribute pigment within the epidermis, and the Langerhans cells (LC). The LC cannot be easily visualized under light microscopy on routine hematoxylin eosin staining and require special stains. They have characteristic and distinct features on electron microscopy.

No specific role was assigned to LC for many years. Several elegant studies have conclusively shown that Langerhans cells are members of the monocyte-macrophage-histiocyte system. They share with them common morphologic and physiologic features.

Like macrophages, LC have surface receptors for the Fc portions of IgG and C3 receptors. They have only a moderate phagocytic potential. Cells in the dermal infiltrate of histiocytosis-X have similar surface markers. LC do not form E rosettes or bear surface immunoglobulins and, therefore, do not resemble lymphocytes morphologically.

Initiation of T-cell reponse is an important function of macrophages. They play an important role in the processing of the antigen and effectively present immunologically active moieties to T lymphocytes. This interaction may be achieved by the generation of a soluble signal or by direct cell to cell interaction. In the latter method, in the first step, a nonspecific binding occurs. During the second step, a binding occurs which is antigen dependent, not easily reversible and eventuates in a proliferation of bound T lymphocytes. Evidence has accumulated to suggest that this macrophage-lymphocyte interaction is regulated and restricted by gene products linked to the major histocompatibility complex (MHC) which are under the control of the autosomal dominant immune response gene. Several experimental studies have shown that these genes code for the Ia antigens present on cell surface. Homology between these structures is necessary for effective cellular interaction.

Recent studies have shown that Langerhans cells bear Ia antigens. Like macrophages, they process antigens, present them to lymphocytes, evoke a blastogenic response and have a stimulating capacity in mixed leucocyte cultures (MLR). Abrogation of these functions by treatment with anti-Ia antisera further substantiates these functions.

These new discoveries on the genetically restricted antigen processing function of the Langerhans cell will add to our better understanding of contact dermatitis while their stimulatory capacity in MLR should have a considerable impact in the field of skin transplantation.

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DNCB—Useful New Compound

DINITROCHLOROBENZENE (1-chloro 2,4-dinitrobenzene) (DNCB) is a simple compound that has become increasingly useful in medicine. It functions as a hapten and consequently becomes a complete antigen when attached to a carrier protein. Approximately 95 percent to 97 percent of the population can be sensitized to it at high concentrations and subsequently develop allergic